REMARKS

Applicants note that all amendments and cancellations of Claims presented herein are made without acquiescing to any of the Examiner's arguments or rejections, and solely for the purpose of furthering the prosecution of the application and to further Applicants' business interests, and without waiving the right to prosecute the amended or cancelled Claims (or similar Claims) in the future.

In the Office Action mailed December 28, 2009, the Examiner issued a number of rejections. Each of the rejections is discussed in detail below.

I. The Claims are Enabled

In the Office Action of December 28, 2009, Claims 1-15 are rejected under 35 U.S.C. §112, first paragraph, as allegedly non-enabled. The Examiner states, "…the specification does not reasonably provide enablement for a method of 1) a method of inhibiting a Th2 cytokine and/or inducing a Th1 cytokine or a method of stimulating an immune response that does not have a result; 2) a method that administers an ODN but does not express the ODN; and 3) a method for treating an inflammatory skin disease that administers the CpG ODN to the subject but not the site of inflammatory skin disease." (Office Action, page 5).

In order to further their business interests and the prosecution of the present application, yet without acquiescing to the Examiner's arguments, and while preserving the right to prosecute the same (or similar claims) in the future, Claims 1 and 7 have been amended to recite an outcome (stimulation of an immune response or inhibition of a Th2 cytokine and/or induction of a Th1 cytokine). Support for this amendment can be found in the specification (e.g., Examples 1-3). Applicants note that expression CpG ODN is not needed because the active reagent for present invention is CpG ODN itself, as recited in the claims. Accordingly, applicants submit that Claims 1-10 are enabled.

Applicants further submit that Claims 11-15 are enabled. Nonetheless, applicants now provide the declaration of Dr. Tae-Yoon Kim, one of the inventors of the present application. Dr. Kim's declaration describes experiments conducted using the methods described in the present application. Section 1 and 2 of the declaration describe the effect of CpG ODN 46-0 via intravenous injection. Section 1 discloses the preventive effect on atopic dermatitis of CpG ODN via intravenous injection in the skin hyperimmune response model induced by the ovalbumin

(OVA) antigen. As shown in Fig. 1-2A (attached to the declaration), the skin lesion of the mouse treated with the CpG ODN and OVA by i.v. injection shows remarkable improvement compared with that of the mouse treated with PBS. In addition, the CpG ODN proves to be more effective as the frequency of injections is increased. Also, as shown in Fig. 1-2B, hyperkeratosis and acanthosis result in remarkable decrease in skin lesions of the mice which were intravenously injected with CpG ODN.

Section 2 of the declaration describes the treatment effect of CpG ODN on the skin hyperimmune response model induced by ovalbumin. As shown in Fig. 2-2A, skin lesions is remarkably improved with the treatment of CpG ODN. H&E staining shows that hyperkeratosis and acanthosis are remarkably decreased (x200) in the lesions treated with CpG ODN. In addition, as shown in Fig. 2-3, the levels of total IgE and antigen specific IgE increased, while antigen levels decreased markedly by the treatment with CpG ODN. As shown in Figs. 2-4, cells which were isolated from a CpG ODN treated mouse did not express IL-4 at all, even when treated with OVA again, thereby showing that CpG ODN has long term effectiveness.

Sections 3 and 4 of the declaration describe the effect of CpG ODN by intraperitoneal injection. Section 3 discloses the treatment effect of CpG ODN on the atopic dermatitis animal model induced by hapten (TNCB) in NClNga mouse. As shown in Fig. 3-2A, the skin lesions of the mouse injected with CpG ODN by i.p. are remarkably treated when compared with that of the control (PBS-treated) group. In addition, H&E staining of the skin lesion sites shows that hyperkeratosis and acanthosis are lessened in the lesions of the mouse injected with CpG ODN as shown in Fig. 3-2B (x200). As shown in Fig. 3-3, the level of IgE remarkably decreases in the 46-0 treated mouse.

Section 4 discloses the effect of CpG ODN by i.p. by inhibiting delayed immune suppressive response, as shown in Fig. 4-2.

Section 5 describes an asthma model for intravenous injection and discloses the effect of CpG ODN on animals in the asthma model. As shown Fig. 5, symptoms of bronchiolitis obliterans of CpG ODN treated group are decreased compared to those of the positive control (Fig. 5-2). Also, CpG ODN remarkably inhibited cell proliferation (Fig. 5-3) and decreased the number of eosinophiles (Fig. 5-4) and the level of OVA-specific IgE (Fig. 5-5). These results demonstrate that CpG ODN described in embodiments of the presently claimed invention have a preventive effect on asthma, which is one of the Th2 type diseases, as well as on atopic dermatitis.

In conclusion, applicants submit that they have provided evidence that CpG ODN can be administrated by topical application and systemic administration such as i.p. and i.v. to control Thl/Th2 immune response balance and to treat a skin disease (see Examples 4 to 7 and enclosed declaration). Administration methods or sites do not affect the effect of the present invention. Accordingly, applicants respectfully submit that Claims 1-15 are fully enabled by the specification, and should be passed to allowance.

II. The Claims are Not Subject to Double Patenting

The Examiner rejects Claim 17 as being a substantial duplicate of Claim 16. Applicants have canceled Claim 17. As such, the rejection is moot.

CONCLUSION

For the reasons set forth above, it is respectfully submitted that Applicants have addressed all grounds for rejection and Applicants' claims should be passed to allowance. Reconsideration of the application is respectfully requested. Should the Examiner believe that a telephone interview would aid in the prosecution of this application, Applicants encourages the Examiner to call the undersigned collect at (608) 662-1277.

Respectfully submitted,

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